

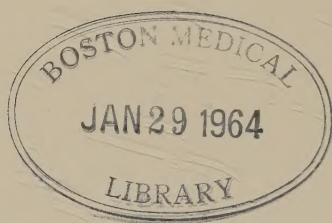
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SERIOUS SYMPTOMS
IN
CHRONIC HEART
DISEASE

HARRINGTON BENNETT MUNROE, M. D.



t. 2784

Doctor Robert Esgood

With regards of
The "Subject"

Much of this
is the history
of the breed of
Hairy

~~Santa Monica~~ Monica
Calif.

Harrison B. Munroe

To
ELLEN HYDE SCOVILL

SERIOUS SYMPTOMS
IN
CHRONIC HEART DISEASE

BY
HARRINGTON BENNETT MUNROE, M.D.



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By HARRINGTON BENNETT MUNROE, M.D.

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PREFACE

Few physicians realize the important changes that have taken place in cardiology during recent years. Wider clinical experience and more accurate records from modern mechanical appliances have relegated older ideas into the realm of the obsolete. And out of the newer knowledge an entirely new point of view has developed with reference to chronic heart disease.

No longer can we truthfully tell our patients that heart disease results from physical strain. Ample evidence exists to prove that a healthy heart is never permanently injured by either hard work or strenuous exercise. Leading authorities are today convinced that the collapse of a damaged heart is attributable to the onslaught of some severe infection and not to overwork.

No longer, moreover, can we base the diagnosis of chronic heart disease on the discovery of the traditionally-important systolic murmur which is best heard at the apex. Experience has demonstrated that a mitral systolic, presenting all the classical characteristics of intensity and transmission, does not necessarily connote structural valvular lesion. In fact it is now generally agreed that an apical systolic, of itself, is seldom of much significance.

Graphic records, obtained from the electrocardiograph and other modern methods, have necessitated a radical revision in our former valuation of heart symp-

toms. In the older cardiology, diagnosis centered on the valves, and symptoms were considered serious largely insofar as they afforded evidence of disturbances in these structures. But in the newer cardiology the emphasis has shifted from the valves to the entire mechanism of the heart. Signs and symptoms are now considered serious largely insofar as they indicate progressive functional inefficiency. Chronic heart disease, in the older conception, consisted *mainly of chronic valvulitis*. Chronic heart disease, in the new interpretation, consists *mainly of chronic carditis*.

We must also readjust our older ideas in relation to the seriousness of cardiac disorders. Pneumonia, until quite recently, held undisputed title as "the captain of the hosts of death." But of recent years pulmonary inflammation has gradually lost this unenviable priority. Today, in this country, the deadliest of all bodily infirmities is chronic heart disease.

Startling evidence of this can be found in any of the larger mortality lists. The Metropolitan Life Insurance Company's reports, covering recent years, show that out of every thousand deaths, over one hundred and twenty-five are due to heart disease, while more than two hundred and fifty are caused by that closely-connected group which consists of cardio-vascular-renal disorders. One can better grasp the significance of these figures by remembering that if this rate continues one out of every four adults now living will die of heart, artery, or kidney disease.

It is imperative that we combat a mortality which is so menacing to middle age. For cardiac and arterial

breakdowns are prone to occur in the forties and fifties, and when far advanced they do not readily respond to treatment. No elixir will rejuvenate the damaged tissues of disabled hearts, and no method has yet been discovered to restore the flexibility of indurated arteries.

Success in lessening the loss of life from cardiac failure depends, in the main, on early and accurate diagnosis. Yet a correct diagnosis at the beginning of breakdown in the cardiac mechanism is not an easy achievement. Every examination of the heart, therefore, should include not only inspection and auscultation but also a careful checking up of the clinical findings by the fluoroscope and electrocardiograph. The complete reports must then be correctly interpreted in the light of modern knowledge.

This book has been prepared to aid the busy practitioner in the recognition, interpretation, and valuation of those outstanding signs and symptoms which, from the new viewpoint, are considered serious in chronic heart disease. It is hoped that the summary here given will prove helpful in the diagnosis of cardiac breakdowns at an early stage, for recent research work has demonstrated the fact that a heart which exhibits any group of the symptoms outlined in the following pages is supplying definite evidence of chronic carditis.

H. B. M.

Los Angeles, July 1, 1928

FOREWORD

The nomenclature of cardiac conditions at the present time is altogether unsatisfactory. Many clinical terms, including "myocardial insufficiency," "hypertensive cardiac disease," "cardiac dilatation and hypertrophy" and "chronic myocarditis" have been employed to designate the numerous disorders usually grouped under the heading of chronic heart disease. These terms, however, are all of them too limited, inasmuch as they lay the emphasis on some particular pathological lesion.

It would be helpful if some definite classification of heart disturbances could be agreed upon and designated by some generally accepted term. But, as Christian points out in the *Oxford Medicine*, "the term used should include in its meaning the possibility of degeneration, cellular infiltration, interstitial connective tissue increase, and, in addition, a functional inefficiency without demonstrable organic lesion." Obviously a term of such inclusiveness will be difficult to find.

Yet it is desirable that we adopt for purposes of convenience some comprehensive designation which, like nephritis in kidney disease, may be used in a general way to designate the group of disturbances which occur in chronic heart disease. In the following pages, accordingly, the term "chronic carditis" has been selected as the most suitable for that purpose.

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The term chronic carditis, although somewhat objectionable, in that it implies an inflammatory condition, serves nevertheless to emphasize the new and important idea that chronic heart disorders are seldom limited to a single structure, such as the valves or the myocardium, but usually involve the entire cardiac mechanism.

Many leading authorities, indeed, are now convinced that a cardiac disturbance sufficiently severe to interfere permanently with the functional capacity of the heart has progressed to such an extent that careful examination will reveal, in addition to signs of cardiac inefficiency, symptoms also of functional disturbances in both the vascular and the renal systems.

Much evidence likewise exists to support the belief that in chronic heart disorders we are dealing with a cardiac-vascular-renal syndrome in which, although the arteries and kidneys show impairment, it is the heart that has borne the brunt of the attack, and consequently it is the heart that displays the most recognizable symptoms.

CHAPTER I

ENLARGEMENT OF THE HEART

Enlargement of the heart may be due to dilatation of the cavities of the heart, or may result from hypertrophy of the heart muscle. This distinction between dilatation and hypertrophy was formerly regarded as a matter of great importance; today it is considered of slight value. To distinguish between these two forms of cardiac enlargement, moreover, is a difficult task. Sir Thomas Lewis of London, one of the best-known authorities on heart disease, frankly acknowledges his failure to make any clear differentiation between dilatation and hypertrophy in living and chronically enlarged hearts. He maintains, likewise, that to determine definitely the comparative degrees of these two forms of enlargement is seldom, if ever, possible.¹

WHAT CONSTITUTES CARDIAC ENLARGEMENT

Considerable differences of opinion exist regarding what constitutes enlargement of the heart. The outside limit for the left border of the normal heart was formerly placed at three and one-half inches from the mid-sternal line. This limit, however, is probably too restricted, for the size of the heart in healthy adults shows great variation. It is safer to assume that when the left cardiac border extends four and one-half

¹Lewis: *The Soldier's Heart and the Effort Syndrome*, (London, 1918).

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inches or more beyond the mid-sternal line (in the fifth interspace) the heart is definitely enlarged. This holds true, again, if the left margin is found in the sixth or lower interspaces.

MEASURING THE SIZE OF THE HEART

The size of the heart can be measured by both clinical and mechanical methods. Clinical methods are perhaps the more important owing to the fact that the necessary apparatus for mechanical measurements is not always available. But it should always be remembered that neither clinical nor mechanical findings are absolutely accurate. A slight degree of error in cardiac measurement, nevertheless, is of little importance, since the enlargement of the heart must be substantial before it can safely be relied upon as a serious symptom of chronic heart disease.

CLINICAL GUIDES TO THE SIZE OF THE HEART

The most reliable clinical guide to the size of the heart is *the position of the left cardiac border*, and this border is best determined from the area of maximal impulse of the apex beat.

The limits of the area of maximal impulse can be outlined by the following method: have the patient standing, with all clothes removed down to the waist line. Place the palm of the right hand on the fourth, fifth and sixth interspaces. Push the fingers well towards the left axillary line, and then move the hand back and forth over the front of the chest wall until the apex beat is clearly felt. Then gradually draw the

hand towards the mid-sternal line until the index and middle fingers are over the apex beat. By palpation with the tips of the fingers, the area of maximal intensity of the apex beat can thus be defined.¹

At times there may be some difficulty in locating the area of maximal impulse, in which case it will be found helpful to have the patient breathe deeply and palpate carefully during expiration. If this method prove unsuccessful, ask the patient to lie on the left side and take deep breaths. In this position examine again during expiration.

When the area of maximal impulse is found, its exact location should be determined by counting the ribs. Note whether it is in the fourth, fifth or sixth interspace and measure the distance from the mid-sternal line.

The position of the area of maximal impulse is the best clinical guide to the size of the heart for, as Lewis points out, the outermost limit of this small area "*corresponds very accurately with the left limit, as depicted by the orthodiagraphic shadow, in hearts of normal size or in hearts slightly or moderately enlarged.*"²

ANOTHER CLINICAL GUIDE TO ENLARGEMENT

In determining the size of the heart some valuable information may be gained by placing the hand over the ribs. In enlarged hearts, one can often feel a clearly-defined movement not only of the ribs but of the lower sternum. A forcible thrust, palpable in sev

¹Heatherly: *Modern Methods in Heart Disease*, (London, 1923).

²Lewis: *The Soldier's Heart and the Effort Syndrome*, (London, 1918).

eral rib spaces, is likewise strongly suggestive of enlargement. Both these movements give to the palpating hand a characteristically sustained sensation.

THE CLINICAL EVIDENCE OBTAINED FROM PERCUSSION

Until quite recent times no examination of the heart was complete without the evidence obtained from percussion. Many examiners claimed great accuracy in outlining the margins of the heart by this method. But these findings, when checked up by the markings of the orthodiagraph, were found to be very inaccurate. Today, leading authorities insist that accurate determination of the size of the heart by percussion is seldom, if ever, possible. Percussion is valuable as a means of making certain that no cardiac displacement exists; but *for ascertaining the left margin of the heart*, reliance should be placed, not on percussion of the cardiac dullness, but on the location of the area of maximal impulse.

Regarding *the position of the right margin of the heart obtained by percussion*, Lewis maintains that it has little, if any clinical value. Furthermore, he holds that when the area of right dullness is found to be greatly increased, such a sign does not indicate enlargement of the right ventricle; it merely provides evidence that the right auricle is engorged.¹ But percussion of the right margin of the heart is not necessary for diagnosing an engorged right auricle. A much more reliable symptom of engorgement of the right auricle is found in the *over-distension of the veins*.

¹Lewis: *The Soldier's Heart and the Effort Syndrome*, (London, 1918).

Take a healthy adult standing at ease and examine the veins. It will be found that all the veins which lie above the level of the base of the heart are collapsed. But if the patient has an engorged right auricle all the veins lying above the level of the base of the heart will be found over-distended. This is a simple test to make, and the results are always plainly visible. Marked distension of the veins not only indicates an engorged right auricle; it also constitutes one of the most trustworthy symptoms of chronic heart disease.

MEASURING THE HEART BY MECHANICAL METHODS

The outline of the heart can best be determined by an examination with the X-ray. The most accurate outline is obtained by a special apparatus known as the orthodiagraph. In this mechanism, the tube of the X-ray is encased in such manner that rays pass through the chest in a small area only. By that process all risk of considerable error is eliminated, and the heart can be measured during a phase of the cardiac cycle. Where the orthodiagraph is not available, a fluoroscopic examination should be made whenever enlargement of the heart is suspected.

THE SIGNIFICANCE OF ENLARGEMENT OF THE HEART

Extensive and permanent cardiac enlargement is one of the most common and most serious symptoms of chronic disease of the heart. When the left border of the heart extends well beyond four and a half inches from the mid-sternal line, a definite diagnosis can be made of chronic carditis.

Many of the older ideas concerning enlargement of the heart are no longer tenable. Until recently it was orthodox to teach that cardiac hypertrophy was a physiological process, and so long as the increase in size enabled the heart to maintain the normal circulation, the condition was described as "*compensation*." When, however, dilatation was superimposed upon this hypertrophy, the process was considered pathological and the heart condition was then referred to as "*decompensation*."

This theory of "compensation" and "decompensation," in the light of modern knowledge, is known to be without validity. In the first place, extensive and permanent hypertrophy of the myocardium is seldom, if ever, a physiological process. In normal hypertrophy, a marked increase in muscular tissue results invariably in the development of a more powerful and efficient muscle. But extensive cardiac hypertrophy does not produce a more efficient heart. Ample evidence exists to prove the statement that a greatly enlarged heart does not stand up under strain and stress as well as a heart of normal size. The large heart frequently fails without warning and under conditions which seem to throw upon it very little added burden.

Recent studies have also demonstrated that dilatation is seldom superimposed upon a chronic cardiac hypertrophy. Cabot of Boston, in an exhaustive summary of over four thousand necropsies carried out at the Massachusetts General Hospital, extending over a period of twenty-five years, showed that two out of every three patients with cardio-vascular disease ex-

hibited some degree of enlargement. And in this long list of autopsies, enlargement of the heart was due in practically all cases to *both hypertrophy and dilatation*. This association of hypertrophy and dilatation, moreover, did not constitute merely a late manifestation in chronic diseases of the heart but existed in both mild and severe cases throughout all stages of enlargement.¹

The permanently enlarged heart should never be looked upon as "compensated." It is more in unison with the actualities to assume that chronic cardiac enlargement is practically always due to the association of hypertrophy and dilatation, and hence that it indicates a degeneration of the heart muscle. Most authorities now believe that a healthy heart never enlarges to any extent—even from the most strenuous exercises. A normal heart muscle has a tremendous amount of reserve to take care of added burdens, and it is only when the myocardium has been damaged by infection or chronic intoxication that it responds to overwork by enlargement.

It must be kept in mind, however, that enlargement of the heart is sometimes nothing more than an acute and temporary condition due to toxemia. The cause may be some known or unknown infection, and with the elimination of the poison the enlargement disappears. This type of acute enlargement, of course, can usually be differentiated from a chronic condition.

¹*Cabot: Facts on the Heart, (Philadelphia, 1926).*

CHAPTER II

DIASTOLIC MURMUR AT THE BASE OF THE HEART

A true diastolic murmur occurs in the interval between the second sound of one heart beat and the first sound of the following beat, that is, in the diastole of the cardiac cycle. The murmur may be heard in the early, middle or late part of this diastole; it may even be heard throughout the entire interval. Frequently a basal diastolic murmur replaces the second sound.

THE CHARACTERISTICS OF A BASAL DIASTOLIC MURMUR

In the early stages of cardiac disease, a diastolic murmur is often heard at the base of the heart, *immediately after the second sound*. This murmur may be scarcely audible, "a faint sigh" or it may be likened to a "short hiss."¹ Sometimes it is so indistinctly perceptible, that it may easily pass unnoticed. At later stages of the disease the murmur increases in length, and instead of occurring immediately after the second sound it may fill the entire period of diastole, often even replacing the second sound. At such stages the murmur is either high pitched and blowing, or loud and whistling. At times it may even be musical.

THE TO AND FRO MURMUR

When a diastolic murmur is heard at the base of the heart there is usually present, in addition, a well-

¹*Mackenzie: Diseases of the Heart, (London, 1925).*

marked systolic murmur. The systolic is heard throughout the entire interval between the first and second sound and generally replaces the first sound.

So, in fully developed disease of the heart, the whole cardiac cycle is filled with murmurs. The combined systolic and diastolic, commonly called the "*to and fro murmur*," replaces not only both normal first and second heart sounds but is heard throughout both the systolic and diastolic intervals.

WHERE TO LISTEN FOR THE BASAL DIASTOLIC MURMUR

Text books in the past have designated the *second right interspace* as the conventional area for listening to the basal diastolic murmur. But this murmur can be heard much more distinctly *along the left border of the sternum, especially about the third interspace*. The murmur will be found to be conducted down the border of the sternum in the direction of the area of maximal impulse.¹

AIDS IN DETECTING A DIASTOLIC MURMUR

A diastolic murmur can best be located at the base of the heart when the patient is examined standing. This murmur should be sought before the patient is allowed to take any exercises. When there is difficulty in detecting this murmur, it will aid greatly to have the patient breathe deeply and then stop breathing after expiration. Examination can be made in this way during the absence of respiration.

¹Lewis: *The Soldier's Heart and the Effort Syndrome*, (London, 1918).

SYMPTOMS ASSOCIATED WITH A BASAL DIASTOLIC
MURMUR

A well-defined diastolic murmur is seldom heard at the base of the heart without other important symptoms being present. The most notable of these symptoms are the following:

- (1) Corrigan's pulse.
- (2) Excessive pulsation in the arteries.
- (3) Capillary pulsation.
- (4) Wide variations in blood pressure.

(1) CORRIGAN'S PULSE

"Corrigan's" or "water hammer" are names given to a peculiar pulse frequently associated with a basal diastolic murmur. The marked characteristic of this pulse is its *collapsing quality*. This collapsing propensity can best be brought out by asking the patient to lift his arm above his head. In this position the palm of the examiner's hand is placed over the front of the patient's wrist. The slapping beat of the radial is distinctly felt. The pulse wave strikes the palm of the hand with decided force—a well defined quick thrust—and then collapses. The sensation obtained while palpating the pulse is that of a very sudden rise followed by an equally sudden fall.

(2) EXCESSIVE PULSATION IN THE ARTERIES

When a marked diastolic murmur is heard at the base of the heart, examination of the arteries generally reveals excessive arterial pulsation. It must be remembered that visible pulsation is frequently seen in the

large arteries of young healthy adults, when, for any reason, the heart beats with considerable force. But there is a clear-cut distinction between the arterial pulsation associated with the forcible beating of a normal heart and the excessive arterial pulsation which accompanies a basal diastolic murmur. In the latter condition, the carotids, especially, will be seen to pulsate abnormally. One can distinctly see the throbbing or swinging of these large vessels in the neck. Excessive pulsation may also be visible in the brachials, radials and femorals. Special interest is attached to the pulsation of the brachials. An examination of this artery in the inner side of the arm above the elbow will show the inward and outward writhing with each heart beat—"the locomotor brachial." This phenomenon, however, is also observed in arterio-sclerosis of elderly patients.

(3) CAPILLARY PULSATION

Another associate of a basal diastolic murmur is visible pulsation in the capillaries. This symptom can be seen by pressing firmly an ordinary microscopic glass slide against the mucous membrane of an everted lower lip. With each heart beat one will notice a delicate flushing of the blanched surface beneath the glass. The same symptom can be observed under the finger nails and can also be brought out by reddening a patch of skin on the forehead and noting the blush which appears simultaneously with the beat of the carotid.

(4) WIDE VARIATIONS IN BLOOD PRESSURE

When a diastolic murmur is present at the base of

the heart, particular attention should be given to an examination of the blood pressure. In no other condition, perhaps, will there be found such wide variations between the systolic and diastolic pressures. The readings on the sphygmomanometer will often show a systolic pressure abnormally high (180 or over) and a diastolic unusually low (60 or under).

THE SIGNIFICANCE OF A BASAL DIASTOLIC MURMUR

A well defined murmur, heard during diastole at the base of the heart, is usually regarded as a symptom of damage done to the aortic valve. A basal diastolic murmur also gives evidence that the functioning of the aortic valve has been so seriously interfered with that the valve no longer holds. Formerly, when this condition existed, *a diagnosis of aortic regurgitation* was made.

This diagnosis, however, is now held to be incomplete. For, according to the new viewpoint on heart disease, the valvular lesions result from inflammatory processes superinduced by some known or unknown infection. And it has been definitely proved that an inflammatory process sufficiently severe to cause damage to the aortic valve (resulting in aortic regurgitation) does not stop at the valve. The inflammation extends not only to the valvular structures but beyond the valve to the entire mechanism of the heart. Accordingly, when a well marked diastolic murmur presents itself at the base of the heart, and especially when it is associated with a basal systolic murmur, *a diagnosis can be made not only of aortic regurgitation but of chronic carditis as well.*

One should seldom, in determining chronic heart disorders, depend upon a single symptom, for serious cardiac impairment practically always exhibits itself by several signs and symptoms. Hence if a "to and fro murmur" is present at the base of the heart, together with a collapsing pulse, with excessive pulsation in the arteries and capillaries, and an abnormal range of blood pressure—if these several symptoms are in evidence there need be no hesitation about a diagnosis of chronic cardiac disease.

If, on the other hand, only the basal diastolic murmur is in evidence without any alteration in the character of the pulse, one can usually feel safe in the conclusion that the disease is slight. Not always, however. It should not be the conclusion when the patient gives a history which discloses one or more attacks of rheumatic fever, or where symptoms of distress appear after light exercises. With a rheumatic history, and with breathlessness, one can be fairly certain that the heart muscle is seriously affected.

In weighing evidence from exercise-tolerance, it should always be remembered that patients who exhibit a basal diastolic murmur alone, or a murmur associated with arterial symptoms, often stand work very well. Men with such symptoms have gone through the grueling experiences of trench warfare without breaking down. But heavy work should never be consciously permitted when chronic carditis of this type exists in even a slight degree, because the cardiac breakdown in these patients, when it comes, is rapid and generally results in total invalidism.

CHAPTER III

DIASTOLIC MURMUR AT THE APEX OF THE HEART

A diastolic murmur at the apex of the heart occurs late in the diastolic interval, that is, immediately before the first sound. Hence this murmur is known as a *late diastolic*, or, more commonly, as a *presystolic*. For the sake of clearness, however, it seems advisable to emphasize the fact that the apical murmur occurs *late in diastole* rather than that it precedes the systole of the cardiac cycle.

THE CHARACTER OF THE APICAL DIASTOLIC MURMUR

The character of the diastolic murmur heard at the apex varies at different stages of heart disease. In early disease the murmur has a rumbling rough sound, usually of short duration. Its distinctive quality, however, is its *crescendo character*. *The intensity of the murmur increases until it ends abruptly in the snapping first sound*. Frequently this loudly-accentuated first sound is mistaken for the second sound of the heart. But this mistake can be avoided by feeling for the apex beat close to the stethoscope with the tips of the fingers.

With advancing disease another murmur develops—*an early diastolic, immediately after the second sound*. At first it may be quite indistinct. Later the two murmurs merge and the entire diastolic interval is filled with sound. The intensity of the combined early and

late diastolic is loud in the beginning, then decreases, then increases to end abruptly in the first sound. Laennec aptly described the sequence of these sounds as the "bruit de rappel"—the roll of drums calling to arms. English authorities prefer the less fanciful description of a presystolic murmur with a full diastolic rumble.¹

With fully developed disease, the crescendo character of the murmur is lost. The entire diastole is still filled with sound but the intensity does not increase to an abrupt ending. This change generally comes on with the onset of a new heart rhythm, such as auricular fibrillation or other serious symptoms of exhaustion of the heart.

THE LOCATION OF AN APICAL DIASTOLIC MURMUR

A diastolic murmur present at the apex is heard best in the area of maximal impulse. This area, as has been said, is normally located in the fifth interspace, three to four and a half inches from the mid-sternal line. The maximal impulse area should first be located, and careful examination should then be made in this region for the diastolic murmur. It is important to remember that the area of maximal impulse of the apex beat of the normal heart is small, only slightly exceeding the area of a circle with a diameter of one inch.

AIDS IN DETECTING AN APICAL DIASTOLIC MURMUR

A diastolic murmur at the apex of the heart is often very difficult to detect and may require several exami-

¹*Heatherly: Modern Methods in Heart Disease, (London, 1925).*

nations for its discovery. Special attention should be given to all patients who present a history of one or more attacks of rheumatic fever. And the search should be unusually thorough when, together with a rheumatic history, one finds a systolic murmur at the apex, present in all positions and not related in any way to inspiration or expiration. Another suggestive symptom is a reduplicated or much accentuated first sound.

In examining for a diastolic murmur at the apex of the heart, auscultation should be carried out first with the patient standing. When a murmur is not discovered in this position, then place the patient on a table and suggest some exercises. Sitting up and lying down for six or eight times may be sufficient but sometimes quite vigorous movements are necessary to make the murmur audible. An important point to bear in mind is the necessity of examining the patient immediately after the exercises, as an apical diastolic murmur may be present for a few moments only.

ASSOCIATED SYMPTOMS WITH APICAL DIASTOLIC MURMUR

When a diastolic murmur is audible at the apex of the heart one can usually hear an associated *systolic murmur*. This symptom, however, is of little significance.

More important is the presence of a sensation known as a *thrill*. A thrill may be defined as a vibration of the chest wall. Laennec called attention to the similarity of this sensation to the feeling conveyed to the hand when placed over the front of the neck of a pur-

ring cat. A thrill at the apex occurs immediately before the apex beat, and runs up to and ceases abruptly with the first sound. It is presystolic in time and recurs with each heart beat.

In palpating a thrill, the palm of the hand should be placed lightly on the chest wall in the area of maximal impulse of the heart beat. Some help can be obtained in locating this vibration by having the patient stop breathing and then examining in the lull of respiration. When the sensation is not palpable, ask the patient to perform some exercises, and then, with the patient lying on the left side, examine carefully the chest wall in the region of maximal impulse.

THE SIGNIFICANCE OF AN APICAL DIASTOLIC MURMUR

A diastolic (generally known as presystolic) murmur heard at the apex of the heart has been regarded as an evidence of diseased mitral valve, and upon this symptom the diagnosis of mitral stenosis has been made. But according to the new cardiac orientation, mitral stenosis is not considered a complete diagnosis. When a diastolic murmur presents itself at the apex of the heart, the question to be determined is whether the infection which caused the inflammation of the mitral valve has or has not extended into the heart muscle. For in both the diagnosis and prognosis of chronic cardiac disease, degeneration of the heart muscle is regarded as much more serious than disturbances of the valvular structures. And in the new cardiology, symptoms which give evidence of myocardial inefficiency are of far greater significance than symptoms arising from valvular lesions.

Leading authorities, today, are convinced that even in the early stages, stenosis of the mitral valve is always accompanied by some degeneration of the muscle. Many cases of mitral disease have their origin in attacks of rheumatic fever, but following these attacks the evidences of stenosis do not manifest themselves till long after the acute fever has subsided. Even an early mitral stenosis may consequently be of several years duration.

When, therefore, a mitral stenosis is present, and particularly a well developed stenosis, the diagnosis of chronic carditis can definitely be made. This for the reason that the mitral disturbance constitutes but a manifestation of a serious infection which has damaged not only the valvular structure but the entire cardiac mechanism.

Fully developed mitral stenosis exhibits itself by a distinct diastolic rumble heard clearly at the apex. This is substantiated when we find, associated with the apical murmur, a well defined thrill, presystolic in time and constant in all positions. Symptoms of even more grave significance include marked enlargement of the heart, congestion of the veins, and the onset of abnormal rhythms. These omens, when present, indicate progressive myocardial degeneration and herald an early and complete exhaustion of the heart.

CHAPTER IV

THORACIC ANEURISM

Two forms of aneurisms occur in the wall of the thoracic aorta. One form may be defined as "a circumscribed or localized expansion or dilatation of the lumen of the artery." The second form may be described as a "pulsating tumor, containing blood, in direct communication with the lumen of the artery."¹

SYMPTOMS OF A THORACIC ANEURISM

A thoracic aneurism may exist with practically no symptoms, and while contained within the thorax it may attain a considerable size before presenting any definite evidence of its existence. Symptoms of a thoracic aneurism, when present, may be classified under five headings:

- (1) Abnormal pulsation
- (2) Diastolic shock
- (3) Thrill
- (4) Dullness on percussion
- (5) Pressure symptoms

(1) ABNORMAL PULSATION

Abnormal pulsation is one of the most significant indications of a thoracic aneurism, and when suggestive symptoms are also present, such as engorged thoracic

¹Norris and Landis: *Diseases of the Chest*, (Philadelphia, 1920).

veins, a very careful examination should be made of the chest wall. For thorough inspection a good light is essential, and the upper part of the chest should be explored from all angles. When the pulsation of an aneurism is visible *in the front*, it is generally seen *in the first and second right interspaces near the sternum*.

Pulsation of an aneurism *in the back* is most frequently observed *to the left of the spine*.

When no external tumor is present, the pulsation may be nothing more than a diffuse, heaving impulse. The area of pulsation, however, is sometimes small and sharply defined. At other times the pulsation may lift a large area of the chest wall with every heart beat. The impulse may best be felt by placing the palm of the hand flat over the suspected region. When this procedure fails, it may be necessary to press the finger tips deep into the intercostal spaces. Abnormal pulsation can at times be detected by placing one hand on the sternum and the other on the spine.

When an external tumor is present it appears as a firm, round swelling which has pushed its way through the chest wall by erosion. Palpation should be carried out by placing the hand over the sac (when the tumor is visible), and pulsation will convey to the hand a slow, heavy, forcible impulse occurring in systole. The distinguishing characteristic of the pulsation of an aneurism, however, is its *expansile quality*. Expansile pulsation is a differentiating symptom of great value in diagnosis, and should be searched for carefully.

(2) DIASTOLIC SHOCK

When a thoracic aneurism is present, a sharp short

diastolic shock is often conveyed to the hand during palpation. This sensation may be of considerable intensity.

(3) THRILL

In palpating an aneurism one may succeed in detecting not only abnormal pulsation and diastolic shock but a vibrating thrill in addition. This thrill, when due to thoracic aneurism, is systolic in time.

(4) DULLNESS ON PERCUSSION

Percussion supplies practically no evidence in deep-seated aneurisms, but in large tumors reaching the chest wall, careful percussion will outline an area of increased dullness. This area usually corresponds to the area of abnormal pulsation. Aneurisms of the ascending and transverse arch of the aorta show dullness in the front, while aneurisms of the descending portion usually show dullness in the left interscapular region in the back.

(5) PRESSURE SYMPTOMS

Pressure symptoms are often the only manifestations of a thoracic aneurism. One of the most important of these pressure symptoms is known as Oliver's sign or tracheal tugging. Osler quotes Oliver as to the correct method of examining for this symptom: "Place the patient in the erect position and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and the thumb and use steady and gentle upward pressure on it. If dilatation or aneurism exist, the pulsa-

tion of the aorta will be distinctly felt transmitted through the trachea to the hand."¹ The tracheal tug consists of a distinct downward tug of the trachea with each systole of the heart and is due to pressure of the dilated aorta on the bronchus. Tracheal tugging is a very reliable symptom of aneurism and is seen especially in aneurism of the transverse arch.²

Another pressure symptom is exhibited by inequality of the radial pulses. The pulse on the affected side may be delayed, diminished, or altogether absent. Pressure on the sympathetic nerves causes inequality of the pupils and pressure exerted on the recurrent laryngeal nerve brings on paroxysmal cough. Particular attention should be paid to a patient who exhibits a peculiar brassy cough of a ringing character. Pressure of an aneurism may also produce hoarseness or loss of voice. Pain, too, may be present, by reason of the pressure on the chest wall or vertebrae. Aneurismal pain may be either mild and transient or severe and persistent. At times anginal attacks occur with pain radiating down the left arm or up into the neck. Sometimes the pain follows the upper intercostal nerves.

DIAGNOSIS BY MECHANICAL METHODS

Whenever an aneurism is suspected, a thorough fluoroscopic examination should be made with the X-ray. Often this is the only method which will give convincing proof of the presence of a damaged thoracic aorta.

¹Osler: *Practice of Medicine*, (New York, 1925).

²Cabot: *Physical Diagnosis*, (New York, 1915).

DILATATION AND SACCULAR ANEURISMS

Two forms of thoracic aneurism are found:

- (1) Dilatation of the Aorta
- (2) Saccular Aneurism

(1) DILATATION OF THE AORTA

An aneurism may consist of "a circumscribed or localized expansion or dilatation of the lumen of the artery."¹ Dilatation of the aorta may range from a slight increase in the diameter of the lumen of the vessel up to an extreme degree of distension. It is met with most frequently in the ascending portion of the arch of the aorta where the diameter at times is increased from the normal 5-5½ cm. up to 9 cm. or more. It is important, however, to remember that dilatation of the aortic arch is seen in many patients suffering from hypertension, nephritis and arterio-sclerosis.

(2) SACCULAR ANEURISM

A true aneurism of the arch of the aorta is not a dilatation but a saccular aneurism. This form of aneurism is defined as "*a pulsating tumor containing blood in direct communication with the interior of the artery.*"¹

THE CAUSES OF THORACIC ANEURISM

Osler describes an aneurism as an accident in connection with disease of the vessel wall, and it is probably true that a saccular aneurism is always secondary to some primary disorder.² The originating cause may

¹Norris and Landis: *Diseases of the Chest*, (Philadelphia, 1920).

²Osler: *Practice of Medicine*, (New York, 1925).

be any of the acute infections, but the most important factor in the occurrence of a thoracic aneurism is *syphilitic infection*. When a thoracic aneurism appears in a patient under forty years of age, syphilis is always the primary disease. In later years, a thoracic aneurism may be either a late luetic manifestation or the result of arterial degeneration.

THE SIGNIFICANCE OF A THORACIC ANEURISM

A thoracic aneurism is a serious symptom, and gives reliable evidence that damage has been done not only to the wall of the artery but to the muscular wall of the heart as well. Since aneurisms are largely syphilitic in origin, the heart shares with the arteries in the results of this infection. The heart muscle may be invaded by the spirochetæ, causing cellular infiltration, fibrosis, and finally necrosis. Or the heart muscle may show late syphilitic lesions frequently involving the auriculo-ventricular bundle with the result that injury to these specialized fibres produces many of the disturbed rhythms, notably heart block.

CHAPTER V

PATHOLOGICAL HYPERTENSION

A clear distinction should be made between hypertension that is physiological and temporary, and high blood pressure that is pathological and persistent. A certain degree of arterial pressure is necessary for life, and this essential pressure is chiefly maintained by the contraction of the heart, the elasticity of the arteries, and the peripheral resistance in the arterioles.¹ Pressure is necessary for supplying blood to the various organs, but this pressure is not maintained permanently at the same degree. Fluctuations of great extent will normally occur under different conditions. Mackenzie mentions numerous records which he made of patients having a systolic pressure of 180 mm. at one examination and of only 120 when examined a few days later. No cause for these variations was found.² Slight emotions or exercise may run the pressure up; but hypertension due to these causes is physiological and temporary; hence it should be disregarded. Pathological hypertension, on the other hand, is more or less persistent and permanent. It is due to some abnormal condition existing within the body.

¹*Barker and Cole: Blood Pressure, (New York, 1924).*

²*Mackenzie: Diseases of the Heart, (London, 1925).*

WHAT CONSTITUTES PATHOLOGICAL HYPERTENSION

Considerable research work has been carried on to determine the normal limits of blood pressure in healthy adults at different ages. Perhaps the best authority is Wiggers, who concludes that in adult life up to middle age the systolic pressure ranges from 110 to 135 millimetres of mercury; and the diastolic from about 70 to 100 mm. (with usual range of from 70 to 90). After middle life the systolic figure is usually a little higher, perhaps by 10 mm., although the diastolic pressure changes little in health.¹ Woley gives the following figures:

Average systolic pressure:

Ages 15-30	122
Ages 31-40	127
Ages 41-50	130
Ages 51-60	132

Barker and Cole maintain that there is no normal pressure but that persistent systolic pressures above 135 (after middle life above 145) are to be looked upon with suspicion. Persistent diastolic pressure above 100 should also be carefully investigated.

Vaquez is of the opinion that hypertension exists when the systolic pressure exceeds 160 mm. in the adult male and 150 mm. in the adult female.²

ASSOCIATED VASCULAR SYMPTOMS

Most authorities now hold the opinion that high blood pressure is largely dependent upon increased

¹*Wiggers: Circulation in Health and Disease, (Philadelphia, 1920).*

²*Vaquez: Diseases of the Heart, (Philadelphia, 1925).*

peripheral resistance, and that this increased vascular resistance is due to changes in the smaller blood vessels.¹

The vascular changes associated with hypertension however, are quite different from the chronic vascular degenerations seen in the condition known as arterio-sclerosis. In the former, the alterations are mainly confined to the smaller arteries and the capillaries, while in the latter the aorta and the larger blood vessels such as the carotids and iliacs, are chiefly affected. In arterio-sclerosis, also, as Christian emphasizes, the blood pressure may be normal or even somewhat sub-normal, although in long continued cases patients usually present the clinical picture not only of sclerosis, but also of hypertension and chronic nephritis.² Much evidence, moreover, exists to suggest the theory that these allied disturbances originate in a common cause.

ASSOCIATED RENAL SYMPTOMS

A close relationship seems to exist between pathological hypertension and disorders of the kidney; but this association is not clearly understood. In many cases increased blood pressure and marked renal disturbances appear to be early associated, while in others the hypertension may exist for a considerable period without any evident renal complications.

It is the general rule, however, that long continued hypertension is followed by signs of renal degeneration, evidenced by such symptoms as albumin and casts in the urine. When the heart, too, shows signs of

¹*Barker and Cole: Blood Pressure, (New York, 1924).*

²*Christian: Oxford Medicine (Nephritis) Vol. III, Part II, (New York, 1920).*

myocardial breakdown, the urinary output is lessened and the usual syndrome of chronic passive congestion with renal insufficiency becomes manifest.

THE CAUSE OF HIGH BLOOD PRESSURE

Many theories have been advanced to explain the onset of pathological hypertension, but the underlying cause is still obscure. Johnson, in 1868, propounded the idea that substances which failed to be excreted by diseased kidneys set up a spasm of the peripheral arteries, resulting in hypertension and vascular disease. Gull and Sutton, in 1872, sponsored the suggestion that disease of the small arteries was primary, and that hypertension resulted from the increased peripheral resistance due to narrowing of the lumen of the diseased vessels.

Neither of these theories, however, explains persistent high blood pressure. For Fishberg has proven by anatomical findings that a general narrowing of the lumen of the small arterioles does not always exist in association with pathological high blood pressure, and it has also been demonstrated that pathological hypertension is not necessarily associated with anatomical changes in the kidney.¹

A SYMPTOM OF AN UNKNOWN DISEASE

Pathological hypertension and associated conditions are commonly degenerations of age. They "milestone the march of senility." High blood pressure, however, occurs also in early or middle life, and may prove to

¹*The Anatomic Features of Essential Hypertension. The Journal of the American Medical Association, June 13, 1925.*

be—as Parkinson suggests—a prominent symptom of an unknown disease. This unknown disease is probably a dual affection of the cardio-vascular and renal systems.

Many authorities are also convinced that these degenerations, whether senile or not, are due to earlier infections. Possibly the trilogy of hypertension, arterio-sclerosis, and nephritis may be said to constitute a syndrome of an undiscovered disorder which some day may take its place in the long list of infectious diseases.

THE SIGNIFICANCE OF PATHOLOGICAL HYPERTENSION

Pathological hypertension may exist for a considerable period without an evident damage to the heart. But pathological high blood pressure is eventually accompanied by serious symptoms of chronic heart disease.

Lewis holds that a persistent high blood pressure of 160 or over, in a young man or woman, is always accompanied by myocarditis. This, also, he claims to be true when the systolic blood pressure is permanently 180 or over in elderly adults. Pathological hypertension, he maintains, is almost always associated in young persons with renal disease, and a thorough analysis of the kidney function should be made to ascertain the extent of the trouble.¹

Cabot, of the Massachusetts General Hospital, concludes from a wide survey of heart disorders that hypertension is the common cause of hypertrophy and

¹Lewis: *The Soldier's Heart and the Effort Syndrome*, (London, 1918).

dilatation in more than three out of every four patients with chronic cardiac disease. He is convinced, moreover, from the hospital records of over a quarter of a century, that long-continued high blood pressure practically always results in cardiac enlargement.¹

¹*Cabot: Facts on the Heart, (Philadelphia, 1926).*

CHAPTER VI

EXTRASYSTOLES

Extrasystoles¹ are premature contractions of the heart muscle due to abnormal impulses. The premature contractions differ from normal heart beats in origin, time and rhythm.

THE NORMAL MECHANISM OF THE HEART BEAT

In order to understand *the disordered action of the heart* it is necessary to have a clear perception of the *normal mechanism of the heart beat*. Primarily, the beat of the heart depends on the functions of cardiac tissue, and cardiac tissue possesses four fundamental functions:²

- (1) Rhythm—the power of originating impulses.
- (2) Conduction—the power of transmitting impulses.
- (3) Irritability—the power of responding to impulses.
- (4) Contraction—the power of changing its shape.

(1) THE POWER OF ORIGINATING IMPULSES

The majority of the cells of the heart muscle are intermediate between the striated and unstriated muscu-

¹The term "extrasystoles" is a misnomer. These contractions are not extra, but premature systoles.

²Wiggers: *The Circulation in Health and Disease*, (Philadelphia, 1923).

lar type, but there also exist in the heart wall many other cells which are much more primitive. Primitive cells possess in a rudimentary form every cellular function, and one of the fundamental functions of cardiac muscular cells is the power of originating impulses.

Primitive heart cells form a very important network, embedded in the posterior wall of the right auricle known as the *sino-auricular node*. Wiggers described the sino-auricular node as a special knot of heart tissue located in the groove called the "sulcus terminalis." It is a club-shaped mass, embedded in the musculature forming the posterior wall of the auricle. This node sends some fibres up to the superior vena cava and ends below, midway between the superior vena cava and the coronary sinus.¹ The *sino-auricular node* possesses in a very high degree the power of originating impulses which stimulate the contraction of the heart muscle. It is not, however, the only centre of origin for these impulses.

Another group of primitive cells, known as the *auriculo-ventricular node*, is situated at the base of the septum which separates the auricles. This node is found at the posterior margin and on the right side. These cells also possess in a highly developed degree the power of producing impulses.

Other portions of the heart, besides these two nodes, may become the centres for originating impulses. In the right auricle the posterior wall of the auricular septum can assume this function. In certain disorders, primitive cells in the ventricular muscle often start im-

¹Wiggers: *The Circulation in Health and Disease*, (Philadelphia, 1923).

pulses. Impulses likewise may arise from cells in the tissues which unite the auricles and ventricles. Normally, however, the impulses producing heart beats originate only in the primitive cells of the sino-auricular node.

(2) THE POWER OF TRANSMITTING-IMPULSES

The cells of the heart tissue not only originate impulses but also possess the power to transmit these stimuli. In the auricles, impulses starting at the *sino-auricular node* travel *from cell to cell* to all parts of the auricles. Impulses are also transmitted in this way from the *sino-auricular node*, situated in the posterior muscular wall of the right auricle, to the *auriculo ventricular node*, located at the base of the septum separating the auricles.

Between the auricle and the ventricles, impulses are carried along *a special and differential set of cells* grouped together into a bundle, known as *the bundle of His*. The bundle of His consists of a peculiar band of primitive muscle cells which begins in the auriculo-ventricular node, passes across the auriculo-ventricular septum, and runs downward and forward reaching the membrano-muscular junction of the inter-ventricular septum. Here it divides into two branches—one of which enters the right and the other enters the left ventricle. Each of these branches lies beneath the endocardium, and, passing down the septum, divides and subdivides, thus forming a system of strands which can be traced over the entire inner surface of the ventricles. The bundle of His (sometimes known as the auriculo-ventricular bundle) is *the only path along*

which stimuli can travel between the auricles and ventricles.

(3) THE POWER OF RESPONDING TO IMPULSES

The cells of the heart tissue are capable of responding to impulses, and this propensity is known as *irritability*. Not all stimuli, however, applied to the heart muscle cells bring out responses. When cells have discharged their store of energy they are incapable of further activity. After a lapse of a definite period, these cells normally acquire a further supply of energy and again become irritable.

The susceptibility of cells to stimuli depends on the accumulated supply of energy. Stimuli, therefore, applied to heart muscle cells *while these cells are in contraction bring on no responses* because the cells have discharged their supply of energy. This is known as the *refractory period or refractory phase*. Stimuli, however, applied to these heart cells, *while relaxing or at rest, bring out responses*, due to the fact that resting cells contain a store of energy and in this condition possess the property of irritability.

(4) THE POWER OF CHANGING SHAPE

The heart muscle cells also have the power of changing their shape. This function, known as *contraction*, is performed when the cells respond to stimuli.

In the normal mechanism of the heart beat, the contraction wave begins at the *sino-auricular node* of the right auricle. This wave radiates from cell to cell, in all directions, until the whole auricular muscle is in contraction. Spreading to the outermost muscle fibres,

the contraction wave finds no further path open to it, and—as Lewis describes it—“dies out”. The auricle then remains quiet until a new stimulus excites it.¹

The stimulus (causing the contracting wave in the auricle), in spreading from cell to cell in all directions, reaches the *auriculo-ventricular node* situated at the base of the septum separating the auricles. From this node the impulse is transmitted through the bundle of His to the ventricles, bringing on a contracting wave in the ventricles. The ventricular wave starts in the cells of the apex of the heart and radiates from cell to cell upward until the whole ventricular muscle is in contraction.

THE ABNORMAL MECHANISM OF EXTRASYSTOLES

Extrasystoles or premature contractions differ from normal contractions of the heart muscle in three fundamental features:

- (1) Origin
- (2) Time
- (3) Rhythm

(1) ORIGIN

Normal contractions, causing the heart beats, always originate in one centre. In the auricles, normal contraction waves begin in the sino-auricular node, and from this point spread out in all directions causing regular auricular contractions. In the ventricles, normal contraction waves follow the impulses brought from the auricles through the bundle of His, and radi-

¹Lewis: *Clinical Disorders of the Heart Beat*, (London, 1920).

ate from cell to cell causing regular ventricular contractions.

Premature contractions, however, are abnormal in origin and may arise almost anywhere in the heart—in the auricles, in the ventricles, or in the tissues uniting these structures. As a rule premature contractions originate in the auricles, and these premature auricular contractions are followed by similar premature contractions in the ventricles. This is due to the rule that the ventricles respond to all auricular impulses whether normal or abnormal. At other times, premature contractions may begin independently *in the ventricles* without being preceded by auricular contractions.

(2) TIME

Extrasystoles also are abnormal in time, due to the fact that the time of preparation required for a *premature* contraction is less than that needed for the preparation of a *normal* contraction.

In the normal acting adult heart the *sino-auricular node* starts the impulse and sends forth the stimulus that causes the normal heart beat. This node, or *pace-maker*, as it is sometimes called, gives forth impulses at a uniform and constant rate, since the *sino-auricular node* is under the combined control of the vagus and sympathetic nerves. Terminal branches of these nerves end in this network of primitive cells, and in health the vagus acts as a curb while the sympathetic serves as a spur to the heart rate.

Under normal conditions, therefore, the rate of the heart beat depends upon the frequency of the impulses discharged from the *sino-auricular node*. Each stimu-

lus originating in this node requires a certain length of time to prepare. The time of preparation is very constant in health, reaching nearly two-thirds of a second, and results in a normal heart rate of approximately seventy-two beats per minute.

In premature contractions, on the other hand, the abnormal impulse requires little time for preparation; consequently the muscle cells are prematurely thrown into contraction before the regular impulse which brings on the normal heart beat is ready.

(3) RHYTHM

Extrasystoles also differ from normal contractions in rhythm.

In the mechanism of the normal heart beat the time between impulses is evenly spaced. Each impulse and each contraction is regularly repeated and belongs to a rhythmical series.

When, however, the normal mechanism is disturbed, a premature contraction of the auricles and ventricles occurs before the regular time, and is followed by a long pause, which is known as the *compensatory pause*. After this long pause the normal rhythm returns. It is interesting to note that the time occupied by the premature ventricular contraction and the compensatory pause is approximately the same as the time taken by two complete cycles of the normal rhythm.¹

THE WORK OF THE EXTRASYSTOLES

The work accomplished by the extrasystoles is small. This is due to the fact that the time taken for the

¹*This, however, does not hold true in the case of a premature auricular contraction.*

preparation of a premature contraction is so much shorter than the time taken for the preparation of a complete normal heart beat. Premature contractions, consequently, have so little force that they frequently fail to raise the aortic valves. But when the valves are lifted, both first and second sounds can be heard on auscultation. Otherwise only the first sound is audible.

THE CHARACTERISTICS OF EXTRASYSTOLES

Extrasystoles are weaker than normal contractions, hence the premature beats may not possess sufficient force to produce a palpable pulse. In that case the pulse is intermittent—a pulse beat at intervals being dropped. On listening, however, at the cardiac area simultaneously with the absent beat, an indistinct premature contraction is heard, followed generally by a very forcible contraction. This is a very important point in diagnosis because it differentiates premature contractions from heart block. With an intermittent pulse, one should always listen to the heart sounds with great care, for with extrasystoles a pulse beat at intervals is missing while *the synchronizing heart beat can be heard*. In heart block, on the other hand, a pulse beat at intervals is missing but at the same time *no corresponding heart beat can be heard*.

Extrasystoles are generally associated with a low or moderate heart rate, and are rarely found in patients who exhibit a pulse of 120 or over per minute. These premature contractions are also unstable and should be looked for during the resting period after exercise. A heart may be perfectly regular before exercise but may

show many extrasystoles shortly after the exercises are completed.

Another important characteristic of extrasystoles is their tendency to exhibit *periodicity or regular recurrence*. With careful palpation one may find that every third beat will be missing at the pulse. At times every second beat is absent. In certain pathological conditions every contraction of the heart may be premature and practically no pulse can be felt.

When an extrasystole replaces every third beat of the heart the condition is known as "coupled beats." This is often a grave danger signal and generally indicates an overdose of digitalis. The warning given by coupled beats should never be disregarded, for if the drug be continued, disaster is likely to result.

THE SYMPTOMS OF EXTRASYSTOLES

Extrasystoles may be present for a long time without other symptoms. Patients with premature contractions complain at times of palpitation or fluttering in the heart, or become conscious of the long pause which generally follows these irregular beats. Especially is this the case when a patient retires at night after a hearty dinner, or after a strenuous day at work or play.

THE SIGNIFICANCE OF EXTRASYSTOLES

Extrasystoles are not of themselves an indication of serious damage to the heart muscle. Premature beats may be temporary and may pass off unnoticed; indeed they may be present for years without any grave cardiac disturbance. Lewis holds that though premature contractions increase somewhat the work of the heart

muscle, yet the added burden is seldom sufficient to cause serious embarrassment. He maintains, nevertheless, that extrasystoles are pathological and that the disturbance exists in the cardiac tissues—an indication of disordered nutrition of the heart.¹

Although premature contractions are themselves of little importance, still in practice one frequently finds this irregularity associated with other and more dangerous pathological conditions. For this reason their presence always demands a thorough search of the heart for really serious symptoms, such as diastolic murmurs at the apex and base, thoracic aneurism, marked enlargement, heart block, etc. When extrasystoles are the only pathological condition present, it is the general opinion of authorities that they are of little significance either in diagnosis or prognosis. An anxious patient may therefore be assured of their apparent harmlessness.

¹Lewis: *Clinical Disorders of the Heart Beat*, (London, 1920).

CHAPTER VII

HEART BLOCK

Heart block may be defined as a disturbance of the normal mechanism of the heart resulting in a delay or blocking of the impulses which pass from the auricle to the ventricle.

DIFFERENT DEGREES OF HEART BLOCK

Heart block manifests itself in three different degrees:

- (1) Delayed Conduction
- (2) Partial Heart Block
- (3) Complete Heart Block

(1) DELAYED CONDUCTION

In the normal mechanism of the heart beat the auricles contract first, and the ventricles a little later. A normal interval of about one-fifth of a second intervenes between the end of auricular contraction and the beginning of ventricular contraction. This interval of about one-fifth of a second represents the time taken by an impulse to pass from the auricle to the ventricle along the conduction bundle of His.¹ When, however, infection damages this pathway the impulses are delayed in their transmission from auricle to ventricle. Consequently the interval between auricular and ventricular contraction is lengthened. This delay in conduction can be shown on an electrocardiogram, but

¹Lamson: *The Heart Rhythms*, (Baltimore, 1921).

clinically it is well-nigh impossible to diagnose this degree of disturbance.

(2) PARTIAL HEART BLOCK

Partial heart block has its beginning in delayed conduction but represents a higher degree of disturbance in the disordered mechanism of the heart. Normally the ventricle is dependent upon the auricle for the stimuli which set up ventricular contractions. These stimuli come through the conducting bundle, and when this bundle is *slightly* damaged the impulses are delayed. When, however, the conducting bundle is *severely* damaged, the impulses are not only delayed in transit but occasional stimuli do not get through at all. When this happens, the clinical signs are "dropped beats."

Dropped beats indicate that the ventricle has not contracted, and the absence of contraction results from the blocking of the stimuli which normally come from the auricle. Missed or dropped beats disturb the regularity of the pulse rate causing pauses of unusual length. Dropped beats also upset the normal rhythm of the heart. For synchronous with the pauses in the pulse-beat *no heart sounds are audible on auscultation at the cardiac area. These silent intervals over the heart* represent dropped heart beats and constitute the most important symptoms in the differentiation between heart block and all other arrhythmias.

In the early stages of partial heart block, only occasional beats are dropped, but as the disturbance progresses a new symptom known as *periodicity* is noted. The dropped beats come periodically, setting up

various ratios between the rates of the auricle and ventricle. The ventricle may miss every fourth beat, sometimes every third beat, or even every alternate beat. This last condition is known as 2 to 1 heart block. As the disease advances, the ventricle may receive only one out of every three or one out of every four impulses from the auricle.

(3) COMPLETE HEART BLOCK

Complete heart block is an abnormal condition of the mechanism of the heart in which, owing to serious damage to the conducting bundle, *no impulses whatsoever are transmitted from the auricles to the ventricles*. The controlling power which the auricles exercise over the contractions of the ventricles is lost. When this occurs, either death results immediately or the ventricles assume the function of pacemaker.

Normally the rate of contraction of the ventricles is determined by the rate of impulses sent out from the sino-auricular node in the posterior wall of the auricle. But in complete heart block the ventricles are completely cut off from the auricles so far as the stimuli are concerned, and hence must originate their own impulses. Fortunately the muscular walls of the ventricles possess this power to produce their own stimuli independent of the auricle. When forced to do so, therefore, the primitive cells of the ventricular walls originate rhythmical impulses. These, in turn, set up regular contractions of the ventricles, usually at the rate of approximately thirty beats a minute. It will be noted that the new ventricular rate is less than half the normal auricular rate.

While the ventricles beat independently at about thirty per minute, the auricles also beat independently at the normal rate of seventy-two per minute. Consequently the heart presents the peculiar anomaly of two distinct rates and two separate rhythms. Furthermore, *the auricular rate of seventy-two per minute* is under the control of the pacemaker (the sino-auricular node) and hence may vary in response to changes of bodily position, exercises, or abnormal conditions such as fevers. But the ventricular rate of thirty per minute goes on regularly, quite independent of these normal or abnormal variations.

DIAGNOSIS OF PARTIAL HEART BLOCK

The earliest symptoms of heart block cannot be clinically diagnosed. Resort must be made to graphic records in order to detect the beginnings of heart block in delayed conduction. But, as the disease progresses, a partial heart block can easily be recognized by the dropped beats. One can detect an interruption in the regular pulse rate by noting therein an occasional pause of abnormal length. Confirmatory evidence is supplied on auscultation by the *silent interval over the region of the heart*.

Partial heart block is a very unstable disturbance. Work or exercise, sufficiently strenuous to raise the heart rate to over 100 or 120 per minute, will bring about a regular pulse. Then later, with rest, the interruptions will return. With many patients in whom 2 to 1 heart block has lowered the ventricular contractions to 40 or so per minute, strenuous exercises will abruptly double the rate to 80 or more. This sudden

jump in the heart beats is generally followed by an equally sudden fall to forty when the patient has had time to rest.

Partial heart block is associated with other cardiac signs, for here again the rule holds that chronic heart disease seldom manifests itself in single symptoms. When partial heart block is permanent, careful examination will reveal other serious symptoms of chronic carditis, besides the obvious disturbance in the rate and rhythm of the heart.

DIAGNOSIS OF COMPLETE HEART BLOCK

When, on examination, the heart beats are found to number less than forty per minute the diagnosis of complete heart block can usually be made. The presence of a regular rhythm, and the absence of acceleration of the heart rate when the patient changes position suddenly or indulges in work or exercise, are important confirmatory symptoms. For complete heart block is one of the few conditions in which no increase in heart rate follows additional work.

Another and perhaps the most important characteristic of complete heart block is the *varying intensity* of the first and second heart sounds produced by the slow ventricular contractions. Evidence also of the more rapid contractions of the auricles may be visible in the pulsations in the veins of the neck. By listening carefully, one may also be able to hear at the cardiac area, indistinct sounds in the lengthened diastole of the abnormally slow heart cycles.

THE SYMPTOMS OF HEART BLOCK

Heart block of the higher degrees brings about a slowing of the rate of the ventricular contractions. This

lowering of the rate, however, does not necessarily interfere with the normal functioning of the heart. If the heart muscle is not seriously damaged, the circulation carries on, even though the heart contracts not oftener than forty per minute. But few hearts in which heart block is persistent have escaped serious damage. On the contrary, the higher degrees of heart block, when persistent, are manifestations of damage which is not only local in the bundle of His but general throughout the musculature of the heart.

Furthermore, some special symptoms follow the extreme lowering of the heart rate, as seen in well advanced heart blocks. When the heart beats are so far apart that several seconds elapse without a single ventricular contraction, the circulation in the other organs, and especially in the brain, is seriously disturbed. And when the heart beats are reduced to twenty or thereabouts per minute from heart block, the patient generally suffers short periods of unconsciousness.

In extreme blocks, where the ventricular contractions cease for 10 to 15 seconds, patients may exhibit what is known as the *Adams-Stokes Syndrome*. The main symptom of this syndrome—besides the abnormal slowing of the pulse—is a convulsive fit, affecting mainly the face and upper limbs. Associated with the convulsion, the patient exhibits intense pallor, stertorous breathing, and rapid pulsations in the veins of the neck. The attack may consist of one seizure, or it may usher in a period of recurring convulsions, often resulting fatally. The convulsions are very prone to occur when a partial block advances to a higher grade, and especially at the time of change from higher grades to complete heart block.

THE SIGNIFICANCE OF HEART BLOCK

Heart block in all of its three degrees, portrays the damage done to the conducting bundle of His. The damaged area is generally located in the main portion of the bundle, and in many cases is the result of syphilitic infection. Disturbances to this portion of the heart muscle is serious, since there exists no other pathway along which impulses can pass from the auricles to the ventricles, and disease of this bundle interferes with the nice co-ordination that normally exists between the auricular pace-maker and the ventricles.

More serious still is the fact that damage done to the conducting bundle, manifested by heart block, is not a local disturbance but forms part of a widespread involvement of the entire heart muscle.¹ Chronic heart block, especially of the higher degrees, should therefore be regarded as one of the serious symptoms of chronic heart disease.

It should be borne in mind, however, that acute or temporary heart block (as distinguished from the persistent type) may occur at various times. This disturbance may follow acute febrile conditions such as rheumatism, diphtheria, pneumonia, typhoid fever, or any of the numerous disorders due to the staphylococci or the streptococci. Acute heart block, manifesting itself during or shortly after fevers, should be regarded merely as a complication of acute infection and hence as indicating the necessity of prolonging the period of convalescence in order to repair the local damage which the infection has done to the heart.

¹*Lewis: Clinical Disorders of the Heart Beat, (London, 1920).*

CHAPTER VIII

PAROXYSMAL TACHYCARDIA

Paroxysmal tachycardia is the term applied to a disturbance of the cardiac mechanism in which the normal rate of the heart is broken by intervals of rapid and regular beats.

THE CAUSE OF PAROXYSMS OF RAPID RATE

In the normal heart, the impulses producing contraction of both auricles and ventricles originate in the network of primitive cells known as the sino-auricular node, situated in the posterior wall of the right auricle. This network or node is the pace-maker of the heart, and in the adult it sends out stimuli at the rate of approximately seventy-two per minute. Each stimulus from this node is transmitted to the auriculo-ventricular node, and from this center is carried along the bundle of His to the ventricles. Each stimulus therefore sets up a rhythmical contraction of the whole heart.

Other primitive cells, however, scattered throughout the muscular wall of both auricle and ventricle may usurp the function of these nodes and may set up an abnormal center of impulse production. At times the insurgent center originates and sends forth stimuli at a faster rate than the normal pace-maker. When this happens, the new center gains control of the contractions of the heart and sets up a heart rate which may be more than double the normal.

In paroxysmal tachycardia, an abnormal center usurps the function of the normal nodes and starts up ventricular contractions which run as high as 220 per minute. This rapid rate, however, is not maintained permanently because the tachycardia occurs only in paroxysms. During these paroxysms the contractions of the heart are not only rapid but regular, and form a rhythmical series. This regularity suggests that the new center which gains control of the contractions is located in one focus, usually outside the node, in the walls of the right auricle.

THE CHARACTERISTICS OF THE PAROXYSMS

Paroxysms of tachycardia are *essentially short or long runs of premature contractions*. These premature contractions occurring in paroxysms, however, form a regular sequence and differ entirely from a number of isolated extrasystoles. Perhaps the most important characteristic of these paroxysms is their extremely abrupt beginning and ending. While one is listening to a heart beating normally at eighty or so per minute, suddenly the rate jumps to one hundred and sixty or more. And after this abrupt beginning the heart maintains the rapid rate regularly and rhythmically, until the paroxysm ends. Then the heart rate drops back with equal abruptness to eighty or thereabouts per minute, and maintains that pace until another paroxysm returns. This sudden rise, and equally sudden drop in the heart rate, is very characteristic of paroxysmal tachycardia.

Another important feature of this condition is the absence of any change in the paroxysmal heart rate

when the patient exercises. In fact the rapid rate in these paroxysms is apparently uninfluenced by any circumstances whatsoever. The heart rates, during such spells, range from 110 to 220; but as a rule the heart contracts between the limits of 160 and 200 per minute. Another distinguishing feature of this condition is that the rate established in one paroxysm is maintained in each and every succeeding attack.

THE DURATION OF THE PAROXYSMS

Paroxysms of tachycardia vary widely in duration. Some attacks last but a few seconds; others are measured in minutes and hours; and still others keep on going for days. The majority of the attacks, however, are limited to a few hours and they rarely, if ever, exceed two weeks. Sometimes the paroxysm consists of only a dozen rapid beats, and then the heart returns to normal. These brief paroxysms may be repeated at short intervals. It is a general rule that frequent attacks are short-lived, while those that occur at long intervals assume a greater duration.

THE SYMPTOMS ASSOCIATED WITH PAROXYSMAL TACHYCARDIA

A paroxysm that lasts any considerable length of time usually shows associated symptoms. At the beginning of an attack the patient may be conscious of some uncomfortable feeling referred to the heart. A fluttering or throbbing may be the main sensation, or some slight or severe pain may accompany the onset. With a long paroxysm the symptoms may be those of severe cardiac exhaustion,—with cyanosis, enlargement of the heart, and engorgement of the veins of the liver

and the lungs. Death occasionally occurs as a result of this heart failure with congestion, but in the large majority of cases a paroxysm ends without fatal results. With the cessation of the attack the heart rapidly returns to its normal size, the congestion disappears, and the regular rate again is established.

During the paroxysm it should be remembered that murmurs which were previously audible are lost. The presystolic of mitral stenosis, for example, one of the commonest of associated murmurs in this disturbance, is inaudible during the attack.

Another important symptom, frequently noticed in paroxysmal tachycardia, is the occurrence of one or more premature contractions immediately following the ending of a paroxysm. These extrasystoles are often helpful in the diagnosis of this form of abnormal rhythm.

THE DIAGNOSIS OF PAROXYSMAL TACHYCARDIA

The diagnosis of paroxysms of tachycardia is often very difficult. Patients may complain of periodical rapid heart action, and these spells of tachycardia, if not present on examination, must often be diagnosed on very little evidence. Rapid heart action may be due to many causes. Normally the heart responds with an increased rate to work, play, emotions and many other factors. Very fast hearts are also the rule in patients suffering from tuberculosis and exophthalmic goitre; but in all such conditions the special symptoms make the diagnosis clear. When, however, one finds on examination a heart rate of one hundred and sixty per minute, but without any definite co-relating cause, the

diagnosis can usually be made of the presence of a pathological rhythm.

This abnormal heart rate, when regular, is generally due to either the one or the other of two disorders:

- (1) Paroxysmal Tachycardia
- (2) Auricular Flutter

When the rate is rapid and regular, running along for a time and then dropping to nearly normal, the new rhythm is usually due to paroxysmal tachycardia. This diagnosis is confirmed if the onset and ending of the paroxysms are abrupt, and if the rapid rate is maintained without variation on change of position or after exercises.

When the rate is rapid, regular, and maintained at a uniform rate for a period exceeding two weeks, the new rhythm can almost certainly be diagnosed as due to auricular flutter.

THE SIGNIFICANCE OF PAROXYSMAL TACHYCARDIA

In an earlier chapter it was stated that isolated premature contractions did not of themselves indicate a damaged heart muscle. But when these contractions occur in an uninterrupted series, causing paroxysmal tachycardia, their significance is of vastly greater importance. Leading authorities hold the opinion that these attacks are evidence of myocardial mischief and can be classed among the serious symptoms of heart disease. The degree of chronic carditis can best be estimated by the extent of the enlargement that takes place during the attack, by the presence of valvular lesions, and by the frequency and duration of the paroxysms.

CHAPTER IX

AURICULAR FLUTTER

Auricular flutter is a term applied to a disorder of the heart in which the normal contractions of the auricles are replaced by rapid and abnormal muscular movements.

THE NORMAL CONTRACTION OF THE AURICLES

In a normally working heart the impulse which starts the contraction of the auricles originates in the sino-auricular node. The contraction begins as a wave which is transmitted from cell to cell in all directions throughout the auricles. Finally on reaching the outermost fibres of the auricular muscle the contraction wave "dies out". Then the auricles rest until another stimulus starts a new contraction.¹

THE MOVEMENTS OF THE AURICLES IN FLUTTER

The movements of the auricles in flutter may be described as "circus contractions".² In these "circus contractions" a wave begins in the auricle and travels rapidly and continuously round a ring which encircles the openings of the superior and inferior vena cava. Impulses are sent off from this abnormal ring, setting up rapid but feeble movements in the auricular muscle. These movements, (known as flutter), may run as high as three hundred and fifty per minute.

Each impulse which starts the auricles fluttering also causes an impulse to be sent on its way to the ventricles, but the ventricles can not respond to such rapid-fire stimuli. Fortunately auricular flutter is almost always

¹Lewis: *Clinical Disorders of the Heart Beat*, (London, 1920).

²Lamson: *The Heart Rhythms*, (Baltimore, 1921).

accompanied by at least "two to one heart block" in the conducting bundle, so that not more than half the auricular impulses reach the ventricles.¹ Consequently, when the auricles are reacting to three hundred stimuli per minute the ventricles contract at a rate of one hundred and fifty or less.

The auricular contractions in flutter are regular and vary but slightly in rate. These rapid and regular movements may be maintained without variations for long periods—often for months and years. The ventricular contractions are also regular as a rule, but irregularity may develop during the transition from one grade of heart block to another.

THE DIAGNOSIS OF AURICULAR FLUTTER

It is generally impossible to diagnose auricular flutter from clinical signs, hence success is achieved only when graphic records of the heart beat can be obtained. But certain symptoms are suggestive. Flutter is a disease of older patients mainly, and when one finds a regular ventricular rate of one hundred and thirty or slightly higher in elderly patients, the probabilities of flutter should be carefully considered. Confirmatory signs would include history of a sudden onset, with disturbances referred to the heart, and accompanied by a faint spell. When the ventricular contractions persist for more than a fortnight at a rapid and uniform rate, and especially when there is no variation in the rate of the beats with exercise or resting—then the diagnosis of flutter can usually be made.

It is important to remember that flutter may also

¹Lewis: *Clinical Disorders of the Heart Beat*, (London, 1920).

occur in paroxysms, and there is no clinical method of differentiating paroxysms of flutter from paroxysmal tachycardia. The maximal rates of these two forms of rapid hearts, however, are valuable guides. Flutter never starts up a ventricular rate of over one hundred and eighty, while in paroxysmal tachycardia the ventricular beats may run as high as two hundred and twenty per minute.¹ Paroxysms of rapid and regular heart beats, lasting over two weeks, should always be considered attacks of flutter.

THE SIGNIFICANCE OF AURICULAR FLUTTER

Flutter of the auricles, with tachycardia of the ventricles, is classed as a serious symptom of chronic heart disease because this condition is practically always associated with damage to the myocardium. Hence the significance of flutter depends largely on the extent of the muscular mischief. When the auricular and ventricular walls are only slightly degenerated, auricular flutter may continue for several years without bringing on any serious breakdown of the mechanism of the heart. Signs of exhaustion, evidenced by angina or congestion, are not usually present. But there is always grave danger that the full number of the impulses which are sent out from the auricles may get through to the ventricles. If this should happen, a ventricular rate might be set up so high that unconsciousness and death would almost immediately follow.

Flutter is one of the disturbances of rhythm which responds well to full doses of digitalis and the prognosis of flutter largely depends on the results obtained after treatment with this drug.

¹*Lewis: Clinical Disorders of the Heart Beat, (London, 1920).*

CHAPTER X

AURICULAR FIBRILLATION

Auricular fibrillation is a disturbance of the cardiac mechanism in which the normal heart beats are replaced by twitching movements of the auricles and irregular contractions of the ventricles.

THE NORMAL HEART BEAT

The normal heart beat consists of an auricular contraction or systole followed by an auricular relaxation or diastole in conjunction with a ventricular contraction or systole and a ventricular resting period or diastole. A nice adjustment exists between the movements of these two divisions of the muscular wall of the heart owing somewhat to the fact that the impulses which cause both auricular and ventricular contractions arise primarily from the same source, that is, from the sino-auricular node. The auricular systole and diastole, together with the ventricular systole and diastole, make up a complete cardiac cycle.

THE HEART BEAT IN AURICULAR FIBRILLATION

In auricular fibrillation, the normal muscular movements of the heart have lost both their uniformity and regularity, and occur in a disorderly manner. Auricular and ventricular systole and diastole no longer follow each other rhythmically, and the heart muscle as a whole neither contracts nor rests. The cardiac mechanism is entirely upset. A series of twitching movements, devoid of regularity or uniformity, usurp the place of the regular movements of the auricles. And instead of the regular contractions and relaxations of the

ventricles, the ventricular muscular movements take place in an altogether haphazard manner. The smooth adjustment which normally exists between the component parts of the cardiac cycle is entirely lost and the heart carries on as though it had broken away from all control.

The serious symptoms of auricular fibrillation, however, are best noted by examining the abnormal movements of the ventricles, for the ventricles bear the brunt of the fibrillating movements of the auricles.

Normally the impulses which bring about the contractions of the ventricles arise with regularity and uniformity in the sino-auricular node in the auricle. But in fibrillation these impulses are supplanted by a host of irregular stimuli which are thrown disorderly into the conducting bundle of His. Fortunately this bundle is seldom intact, because a considerable degree of heart block practically always exists in association with auricular fibrillation. As a consequence the ventricles seldom receive more than half the auricular impulses. These that do get through, however, upset not only the rate but also the rhythm and uniformity of the ventricular movements.

As a result of this bombardment of the ventricles by abnormal stimuli thrown through the conducting bundle, the ventricular contractions become rapid and irregular. At times, as often happens, when the auricles are fibrillating at three hundred per minute, the ventricles will respond with a rate of one hundred and fifty contractions. Ordinarily the ventricular rate in fibrillation exceeds one hundred per minute.

The rhythm, too, of these contractions is markedly

irregular. This irregularity is so characteristic that some authorities have described this disturbance as "an irregular irregularity."

Not only the rate and the rhythm but also the uniformity of the ventricular contractions is lost. One or two fairly full contractions may occur, but these are followed by a run of several feeble beats. The contractions give the impression of being crowded together and jumbled up. Many of the ventricular contractions are not of sufficient strength to create arterial pulsation, and when the pulse beat is missing, on listening over the heart area the second sound is inaudible. Otherwise both sounds are heard.

Murmurs may be present at times, but these generally are lost, especially when the rate is very rapid. It should be remembered that although auricular fibrillation is closely associated with mitral stenosis the characteristic presystolic murmur of this valvular lesion changes in character or altogether disappears with the onset of fibrillation.

SYMPTOMS ASSOCIATED WITH FIBRILLATION

Auricular fibrillation is always associated with other symptoms of chronic heart disease, because a damaged heart muscle exhibits many signs besides those of disordered rate and rhythm. Careful examination will reveal breathlessness, cyanosis, enlargement of the heart, and other manifestations in the long list of symptoms which follow on the trail of heart failure with congestion. Chronic myocardial degeneration accounts for most of these signs, but fibrillation may of itself greatly augment their severity. Usually patients suf-

fering from fibrillation display a degree of breathlessness and exhaustion far more severe than would be accounted for by a damaged heart muscle alone. Even a short walk brings marked cyanosis and exhaustion. Both of these symptoms are probably the direct result of the increased load thrown on the myocardium by the rapid and disorderly action due to fibrillation.

THE DIAGNOSIS OF AURICULAR FIBRILLATION

The diagnosis of auricular fibrillation can be made with great certainty by an examination with graphic records of the heart beat. But most cases of fibrillation can be discovered without the aid of an electrocardiogram. Clinically, the most important guides are the outstanding characteristics of the ventricular contractions. When one examines a heart and finds the ventricular movements markedly irregular, and exceeding in rate one hundred and twenty per minute, the diagnosis of fibrillation can usually be made.¹

Irregularity and rapidity, however, occur from other causes, for example, from premature contractions. In practically all cases where the heart movements are irregular and rapid the effect of exercise gives valuable information. One will observe that when a patient with premature contractions takes exercise, and thereby brings on a considerable increase in the heart rate, the irregularity due to the extrasystoles disappears.

The irregularity due to fibrillation, however, becomes even more irregular when the heart rate is increased by exercise. This also holds true when the heart beats faster from change of position, emotions, or

¹*Lewis: Clinical Disorders of the Heart Beat, (London, 1920).*

fevers. Probably the chief outstanding characteristic of the irregularity of fibrillation is its persistency, for at no time does the fibrillating heart return to normal rhythm. Fibrillation, once begun and left untreated, continues without intermission until the heart muscle gives out from complete exhaustion.

THE SIGNIFICANCE OF AURICULAR FIBRILLATION

Auricular fibrillation is one of the most frequent and most serious symptoms of heart failure. Lewis and other authorities are convinced that more than half the patients admitted to the hospitals suffering from cardiac failure exhibit this disorder. Usually the onset of fibrillation gives evidence of a heart so badly damaged that life cannot be maintained for more than a few years at the longest.

The seriousness of fibrillation can best be judged by the ventricular rate, for the disordered auricles cause much less embarrassment to the heart than the rapid and irregular ventricular contractions. When the ventricles continue to contract at a rate as high as one hundred and twenty or over, the prognosis is poor. Fibrillation causing ventricular rates of one hundred and forty or one hundred and fifty forecast disaster within a short time. A damaged heart muscle is unable to withstand such an excessive burden.

Fibrillation, together with flutter, are disturbances of the heart which frequently respond well to digitalis or strophanthus, and the prognosis will largely depend on the reaction which follows proper treatment with these drugs.

CHAPTER XI

ALTERNATION

Alternation is a disturbance of the mechanism of the heart in which the force of the beats is alternately strong and weak.

THE MECHANISM OF THE HEART IN ALTERNATION

In the normal heart the ventricles contract with regularity and uniformity, thereby throwing into the arteries an equal quantity of blood with each systole. But in the disorder known as alternation the uniformity of the heart action is upset. The ventricles contract regularly, but a greater and lesser amount of blood is sent into the arteries at alternate beats. This alternation in the work of the ventricles is supposed to be due to the fact that in some contractions not all the cardiac muscular fibres fulfill their complete function.

THE DIAGNOSIS OF ALTERNATION

Alternation cannot be diagnosed by clinical methods, for the variation in the strength of the contractions of the ventricles does not create sufficient difference in the intensity of the cardiac sounds to be detected by the ear. Examination of the pulse in alternation is also not satisfactory because the variations in the force of the pulse can seldom be picked up by palpation. Hence the diagnosis of alternation must be made by instrumental methods.

Perhaps the best plan to determine the presence of alternation is as follows: push up the pressure in the arm band of the sphygmomanometer until all the beats are obliterated. Then slowly relax the pressure until the sounds begin to reappear. Lessen the pressure very gradually and strong beats only will first be heard. The intervals, however, between these strong beats will appear longer than normal. Then, by slowly relaxing the armlet, not only the stronger but the weaker beats can be heard. Careful examination will reveal the fact that the number of the strong beats first heard after relaxing the pressure forms but one-half of the total pulse rate.

Alternation is easy to overlook. But this symptom should be sought for with great diligence in all cases where the heart rate is very rapid. Likewise it should be looked for when the rate is normal in older patients with hypertension, arterio-sclerosis or disorders of the kidneys. Special attention should be directed towards detecting this symptom in aged adults in whom premature contractions are found to occur at frequent intervals.

Alternation is a variable symptom. In some patients the alternate strong and weak beats are present persistently. In many others alternation occurs only during certain periods. Alternate strong and weak contractions frequently occur shortly after extrasystoles. At such times, however, alternation may be wrongly diagnosed unless it is remembered that a premature contraction is generally followed by a contraction of abnormal strength. The heart throws into the arteries, after the long pause which succeeds an extrasystole, a

larger amount of blood than during a normal contraction. The first beat of the heart, therefore, following a premature contraction should be ignored but the following beats are to be watched carefully. Frequently alternation is detected only in these few cycles.

THE SYMPTOMS ASSOCIATED WITH ALTERNATION

Alternation is a serious symptom of heart disease, and, like all other serious symptoms, is never found alone. Chronic heart disease evidences its existence by many signs—breathlessness, hypertension, enlargement of the heart, diastolic murmurs, or dangerous exhaustion symptoms such as angina pectoris. Examination may reveal any of these symptoms associated with alternation. These signs, however, are due to the chronic carditis which is always co-existent when alternation is present.

THE SIGNIFICANCE OF ALTERNATION

Alternation, of itself, is one of the most unmistakable symptoms of a fast failing heart. Its seriousness should never be minimized. When associated with other symptoms of chronic carditis the outlook for the patient is very grave, with little hope of recovery.

CHAPTER XII

CARDIAC EXHAUSTION SYNDROME

Cardiac exhaustion syndrome is a term applied to the group of signs and symptoms which herald a failing heart.

A failing heart may present either one of two clinical pictures—viz:

- (1) Cardiac exhaustion syndrome with congestion.
- (2) Cardiac exhaustion syndrome with pain.

(1) CARDIAC EXHAUSTION SYNDROME WITH CONGESTION

In the great majority of patients with chronic heart disease the earliest, foremost, and most persistent symptom of cardiac exhaustion is *breathlessness*.

Difficulty in breathing may arise from many causes but the dyspnoea of cardiac origin exhibits several very definite characteristics. In the first place, true breathlessness, due to exhaustion of a damaged heart, *never exists except in association with cyanosis*. Some degree of cyanosis is always evident with cardiac dyspnoea. This point cannot be too strongly emphasized, for it distinguishes breathlessness of chronic heart disease from dyspnoea due to all other causes. Lewis emphatically states that there is no exception to the rule that when a patient, *at rest in bed*, exhibits breathlessness without cyanosis, the dyspnoea cannot be caused by chronic heart disease.¹

¹Lewis: *The Soldier's Heart and the Effort Syndrome*, (London, 1918).

Difficulty in breathing of cardiac origin is also accompanied by marked facial expressions of anxiety. A patient with well developed cardiac dyspnoea brings every muscle of respiration into action and strives desperately to obtain a sufficient supply of air. Speech is interrupted after every few words to allow time to gasp for breath. Breathless patients cannot stop breathing even long enough to swallow nourishment without exhibiting evidences of distress. At times the air hunger is so extreme that a sufferer is unable to breathe while lying down and escapes suffocation by being constantly propped up. This form of dyspnea is known as orthopnea.

Associated with breathlessness and cyanosis in cardiac exhaustion syndrome, one will generally discover *over-distension of the veins*. This sign is of very great importance foretelling, as it always does, *the onset of myocardial breakdown with congestion*.

In every examination of the heart a very careful survey of the veins should be made. Notice the veins in the arms when the patient is lying in bed. Normally these vessels will be distended when the arms are resting at a lower level than the base of the heart. But when the arms are raised, the veins should immediately collapse. If, however, the veins in the uplifted arms remain engorged then one can be certain that the venous pressure is abnormally increased.

Examine the patient also in the erect position. While a patient is standing erect, all the veins above the level of the heart should be entirely collapsed. In this position these vessels should show neither distension nor venous pulse with the exception of the large veins in

the root of the neck in which slight venous pulsation is sometimes visible. When these vessels remain full and distended while the patient is standing, the fullness and distension plainly indicate that the venous pressure is increased. And it cannot be too strongly emphasized that this increased venous pressure, evidenced by over-distension of the veins, is one of the outstanding signs of an exhausted heart and displays in no uncertain terms the oncoming of serious cardiac failure.

These three important symptoms—*breathlessness*, *cyanosis* and *over-distension of the veins*—constitute the outstanding trio of what may be called cardiac exhaustion syndrome, indicating collapse of the heart with congestion. This form of myocardial breakdown also presents many other congestive symptoms, including enlargement of the heart, crepitations in the lungs, swelling of the liver, together with evidences of serious kidney impairment exhibited by edema, ascites, and great reduction in the urinary output. Many patients, in addition, show gross irregularity of the heart rhythm due to the onset of auricular fibrillation.

(2) CARDIAC EXHAUSTION SYNDROME WITH PAIN

Heart failure may also present a clinical picture in which pain predominates. The cause of the predominance of pain over all other symptoms in this form of myocardial breakdown is not clearly understood. It is known, however, that pain is a danger signal thrown out by a damaged heart when the heart muscle is overworked and exhausted. This form of cardiac failure is apparently most readily exhibited when for any rea-

son the coronary vessels are diseased, thereby interfering with the supply of blood to the muscular walls of the ventricles.

True cardiac angina possesses many characteristics. This form of pain seldom appears in youth but is found mainly in middle-aged and elderly patients. Perhaps the most important feature of this type of pain is the nature of its onset. Angina pectoris, except in very severe cases, practically never occurs while the patient is at rest unless the resting period has been preceded by some physical exertion. In the early stages of this disorder, hill climbing or severe strain may be necessary to induce an attack. Later, however, a short walk or even a few steps may bring on a severe spell. Patients suffering from attacks of angina are most prone to suffer when work or exercises are undertaken immediately after a meal.

Other important characteristics of this form of pain are the extreme anxiety and the acute sensation of impending disaster which accompany anginal attacks. Many patients, too, complain of a severe constriction of the chest. Attacks vary greatly in duration, some lasting but a few seconds, while others continue for many minutes or even longer. Relief is experienced quickly as a rule by complete rest, though patients frequently complain of a feeling of great exhaustion for some time afterwards.

True angina begins in the region of the sternum and radiates to the neck and arms. This distribution of pain, however, is not a characteristic of angina alone. A better guide is the complaint of a patient that the pain is *most severe across the sternum*. Tenderness of

the skin, together with hyperalgesia of the muscles, frequently accompany the pain and may last long after the attacks are ended.

Disturbances of digestion frequently accompany anginal pain and many attacks are wrongly diagnosed as "acute indigestion." When the substernal pain is persistent and unrelieved by rest the probability of blocking of a coronary artery with myocardial infarction must be carefully considered.

Angina pectoris, and associated symptoms constitute a definite and serious syndrome that herald heart failure from exhaustion and clearly indicate a diagnosis of chronic carditis. Unless the condition is recognized early and precautions adopted to prevent overtaxing the heart muscle, the prognosis in this form of cardiac exhaustion is always serious.

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